Trial Testimony Designations for:
In Re: W. R. Grace & Co., et al.
(U.S. Bankr. Ct., Dist. of Delaware, Case No. 01-1139)
March 26, 2008

Deposition Designation Key

Arrowood = Arrowood Indem. Co. f/k/a Royal Indem. Co. (Light Green)

BNSF = BNSF Railway Co. (Pink)

Certain Plan Objectors "CPO" = Government Employees Insurance Co.; Republic Insurance Co. n/k/a Starr Indemnity and Liability Co.; OneBeacon America Insurance Co.; Seaton Insurance Co.; Fireman's Fund Insurance Co.; Allianz S.p.A. f/k/a Riunione Adriatica Di Sicurta; and Allianz SE f/k/a Allianz Aktiengesellschaft; Maryland Casualty Co.; Zurich Insurance Co.; and Zurich International (Bermuda) Ltd.; Continental Casualty Co. and Continental Insurance Co. and related subsidiaries and affiliates; Federal Insurance Co.; and AXA Belgium as successor to Royal Belge SA (Orange)

CNA = Continental Cas. Co & Continental Ins. Co. (Red)

FFIC = Fireman Funds Ins. Co. (Green)
FFIC SC = Fireman Funds Ins. Co. "Surety Claims" (Green)

GR = Government Employees Ins. Co.; Republic Ins. Co. n/k/a Starr Indemnity and Liability Co.

Libby = Libby Claimants (Black)

OBS = OneBeacon America Ins. Co. and Seaton Ins. Co. (Brown)

PP = Plan Proponents (Blue)

Montana = State of Montana (Magenta)

Travelers = Travelers Cas. and Surety Cos. (Purple)

UCC & BLG = Unsecured Creditors' Committee & Bank Lenders Group (Lavender)

AFNE = Assume Fact Not in Evidence
AO = Attorney Objection
BE = Best Evidence
Cum. = Cumulative
Ctr = Counter Designation
Ctr-Ctr = Counter-Counter
ET = Expert Testimony
F = Foundation
408 = Violation of FRE 408
H = Hearsay
IH - Incomplete Hypothetical
L = Leading
LA = Legal Argument
LC = Legal Conclusion
LPK - Lacks Personal Knowledge
LO = Seeking Legal Opinion
NT = Not Testimony
Obj: = Objection
R = Relevance
S = Speculative
UP = Unfairly Prejudicial under Rule 403
V = Vague

UNITED STATES BANKRUPTCY COURT
DISTRICT OF DELAWARE

IN RE:	.	Case No. 01-1139 (JKF)
W.R. GRACE & CO., et al.,	.	USX Tower - 54th Floor
	.	600 Grant Street
	.	Pittsburgh, PA 15219
Debtors.	.	
	.	March 26, 2008
. . . . .	.	8:38 a.m.

TRANSCRIPT OF TRIAL
BEFORE HONORABLE JUDITH K. FITZGERALD
UNITED STATES BANKRUPTCY COURT JUDGE

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Proceedings recorded by electronic sound recording, transcript produced by transcription service.

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proceed.

THE COURT: All right. Dr. Anderson.

COURT CLERK: Please stand and raise your right hand.

DR. ELIZABETH ANDERSON, DEBTOR'S WITNESS, SWORN:

COURT CLERK: You may be seated. Please speak into the microphone.

DIRECT EXAMINATION

BY MR. BERNICK:

Q Good morning, Dr. Anderson. We apologize for --

(Adjusting microphones)

Q We apologize for the delay in the elevator and then making you sit through scheduling matters, but we'll get to your testimony now.

Could you tell the Court briefly what it is that you are here to address this morning?

A Yes. Your Honor, I am here to address the essential question of whether exposures to Grace products have caused the diseases that the claimants have introduced.

Q Okay. With that as an introduction, could you tell us a little bit about your educational background and we would show at this point demonstration 2264. Go ahead, Dr. Anderson.

A Yes. My academic training began at the College of William and Mary where I was a pre-med student planning to go to medical school and my fourth year I decided to not do that, but I had equivalent training in biology and chemistry and chose

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chemistry as my major. I was solicited by fellowship to go to the University of Virginia to continue in the training and organic chemistry, mechanistic organic chemistry, where I completed my Masters Degree work. And during that time I taught the pre-med sections of the laboratories in organic chemistry, and continued my academic training under a Defense Department fellowship which dictated that I do my research at a military base in concert with an academic institution. Completed by Ph.D. work in 1970.

Q Thank you. The Court has heard about risk assessment already in this trial and, indeed, heard very specifically from Dr. Joseph Rodricks. Do you know Dr. Rodricks?

A Yes. Dr. Rodricks and I were colleagues as early as the mid-70's. He was the functional head of the risk assessment activities at the FDA, Food and Drug Administration and I was the counterpart at EPA.

Q How far back, and maybe you've already identified that, but how far back do you go in the field of risk assessment and I don't mean to ask embarrassing questions, but it's designed to find out what the extent of your background is.

A Yes. I go back to origins of the first applications of risk assessment, to judgments about environmental agents, incorporating for the first time in 1975, the concepts of exposure, past, current and future exposures, to understand the potential for disease causation.

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Q Did your experience include a series of years spent at the EPA, the Environmental Protection Administration?

A Yes. As soon as I was released from my Defense Department fellowship obligations, I went directly to EPA in its first year of operation.

Q I'm going to show you --

A In 1972.

Q I'm sorry. I want to show you demonstrative 2265 and ask you whether that would help you go through your activities at the EPA relating to risk assessment?

A Yes. And, in the early years, I had begun working with the then administrator Bill Ruckelshaus on a series of issues involving carcinogens. It was a period of time in the nation's history where there was thought to be an epidemic of cancer caused by environmental agents. So, the focus at EPA in 1971 was on suspect carcinogenic agents. We worked on a series of pesticides, became aware of a series of air pollutants where there were tumors in animals or humans and we had pursued a policy of zero tolerance because the application of safety factors and threshold levels for safety for carcinogens had not been accepted and the origin of that policy came form the Food, Drug and Cosmetic Act, the Delaney clause, which it said if there were tumors in animals or humans there was no tolerable risk.

Trying to follow that policy at EPA had not worked and I

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was asked to direct the first committee to address a functional cancer policy. That was in the fall of 1975. I was the executive director of that committee. We reported at the first use of risk assessment and risk management that had ever been reported or adopted by any agency in the United States.

That policy called for, for the first time, if you can imagine, exposure assessment, does response modeling, in addition to what had been done in the past. The concept of evaluating how likely an agent is to be a carcinogen based on human data supplemented by animal studies. These first guidelines called for a systematic approach to evaluating the weight of evidence and exposure and likelihood of risk to human populations. It also called for creating a group within the agency to carry out these responsibilities.

I founded and directed that group, it was called the Carcinogen Assessment Group and rapidly, after the success of that group, I founded the Exposure Assessment Group, the Productive Effects Group, and the expanded office for all of the risk assessment activities. I directed that officer for ten years and I am co-author of EPA cancer policy with the then administrator Russell Train. It was published in the Journal of the National Cancer Institute. We performed hundreds of risk assessment. I co-authored those risk assessments during that period.

Q Thank you. I want to ask you about turning to

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demonstrative 2266. There's been a reference made, I believe, by Dr. Rodricks, concerning the red book. Could you tell us about the red book and what your involvement, if any, in the red book was?

A Yes. The EPA experience had formed a lightening rod for discussions about using dose response extrapolations, exposure assessment and estimation of risk for public policy decisions. It was one of the major, I would say, stimulus, for the convening of the National Academy's Committee to address the appropriateness of these policies, could risk assessment and risk management work on a broader basis, and by that time my group at EPA had published more than 150 risk assessments. So, we were very far down the road. This committee and this publication, to which I was an advisor and visited many times because our work was one of the cornerstones of their deliberations, is the landmark book that's established the paradigm for risk assessment. It's cited all the time, cited by parties and interested organizations all over the world and in many subsequent National Academy documents.

Q Thank you. Turning to demonstrative 2006, which is also a demonstrative that we used in connection with Dr. Rodricks testimony, could you tell us briefly how it is that the science of risk assessment has evolved, particularly in the last two or three decades?

A I guess I can. I often think of risk assessment as

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originating with Koch's principles. These principles sought to answer questions of causation for infectious disease, isolating the organism form the host, cultivating the organism and then reinserting it in the host and if the host responded to the original disease, causation was established.

Also, going on pre-1964, in the public health regulatory communities, were these simplistic safety factor approaches that established no observed effect levels or other means of establishing a level that was regarded as safe and in the scientific community, there were early observational studies we call case reports in epidemiology that addressed the results that some medical doctors had diagnosed in their patients, certain diseases that might be associated with occupational exposures.

By 1964, we were seeing several different things happening. One of the critical events in 1964 was the publication of the Bradford Hill criteria which for the first time gave a real structure to what was expected of epidemiology studies, if they were going to establish causality between the agent and the disease and these principles addressed consistence across the studies, the temporal issues and other issues.

These principles promptly became useful, as I understand, to the interface of judicial use, which is not my field, but I understand that structure was useful. But they were useful in

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other ways, because they stimulated the conduct of structured epidemiology studies. So, studies became more structured, they became more useful in many different ways. Of course, human data still remain the best source of information. But following the regulatory approach of what I call the Public Health Agency approach, during the 70's is just what I've discussed. We've encountered the situation with suspect carcinogens and the rejection by the scientific community of just willy-nilly applying safety factors. And, so, the quantitative based risk assessment process was established, but here were see a divergence between what Public Health Agencies were doing to carry out their missions to be preemptive and protective and what was really called for in establishing causality. So, it's been an interesting period and interesting that I've been able to participate in a good deal of it.

Q Well, let's talk a little bit about that divergence. I know we're going to return to it later, but since you've mentioned it, what exactly was the divergence that you just referred to that began to evolve in the 1970's?

A Yes. The process of risk assessment that was defined by EPA in 1975 simply sought to answer two questions. How likely is an agent to cause disease or if a disease is already caused, what is the dose that -- what is the circumstance that might have caused the disease or if a disease is -- if a dose is current, would you expect that there might be impacts on

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populations in the future.

In that case, the Public Health Agencies essentially have to fill data gaps and the red book that I mentioned earlier details those gaps and calls them inference judgments. So when, for example, the EPA did their risk assessments when I was there, we followed guidelines and said where the science stops we will fill the gaps with very precautionary assumptions. So, we were seeking to establish plausible upper bounds on risk, recognizing that the risk could be considerably less, even approaching zero, but to make decisions to preempt and protect the public. It's a very different process from establishing causality which is, as I understand, causality needs to be science based and not inference judgment based.

Q Okay. And I know that we're going to return to that. Turning to slide, or demonstrative 2267, have you been involved in developing literature in the field of risk assessment?

A Yes, I have. I have published, I have participated in founding the leading society in risk assessment. I served as its president from 1984 to 1985. I have held other posts in that society, but I think the most exciting one has been as editor and chief of the Journal, Risk Analysis and International Journal which I think is undeniably the leading journal in the field of risk assessment.

It has a world wide circulation of 4,000, it goes to about 80 countries and its board and its editorial staff are made up

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of scientists from the academic communities, governmental bodies, and international representatives in the private sector. It is ranked high in the impact factor and it's also ranked very high in the social science citation index. We've been as high as number two of 65 journals, I believe, within the last six years and as high as number four, more recently listed in the Interdisciplinary and Mathematics citation index.

MR. BERNICK: Your Honor, at this time, we would proffer Dr. Anderson as an expert in the field of the risk assessment of toxic agents or potentially toxic agents, including specifically asbestos.

MR. MULLADY: Brief voir dire, Your Honor?

THE COURT: Yes.

VOIR DIRE EXAMINATION

BY MR. MULLADY:

Q Good morning, Dr. Anderson.

A Good morning.

Q Your doctoral degree is in organic chemistry, is that correct?

A Yes. Mechanistic organic chemistry.

Q Your expertise in risk assessment, as I understand it form your CV, has been in the context of regulatory and legal matters, is that right?

A I think it's in the scientific context of evaluating multi disciplinary information to address issues of how likely an

agent is to cause disease, to address issues of dose response characteristics, that is, at what level could that disease occur and then to address issues of exposure and then to wed the two, to address the essential questions of how likely is there to be an association between that exposure and disease.

Q May I see 433 please? Exhibit 433 on the screen here is a copy of your CV which I believe you appended to one of your expert reports in this case. And I was reading from the second sentence in the first paragraph which says, she specializes, referring to you, in risk assessment as a basis for addressing the complex problems that arise in the context of regulatory and legal matters, related to health and the environment for national, international companies and governments. Is that accurate?

MR. BERNICK: Your Honor, I don't believe that's proper impeachment.

THE COURT: I --

MR. BERNICK: There'S nothing that's inconsistent about that.

THE COURT: This witness can answer the question if it's coming from her's. I'm not sure it's inconsistent, but I'm sure the witness can answer this question.

Q Is that an accurate statement of your credentials, ma'am?

A Well, I don't think any single declarative sentence can accurately reflect my credentials. I think this sentence

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reflects some of what I've done.
Q Now, you have no formal medical training, is that right, ma'am?
A I have not been to medical school, if that's what you're asking me. No.
Q Yes. You also have no legal training, correct?
A Correct.
Q And, you're not an industrial hygienist, right?
A I am not an industrial hygienist.
Q And you're not a bio-statistician, correct?
A I've had courses in statistics. I have used statistics in my work, but I would not say I'm a bio-statistician.
Q Okay. And, as I understand it, none of the risk assessments that you've done, that have dealt with asbestos, have been published in a peer reviewed journal, is that right?
A That's wrong.
Q That's wrong? What risk assessments on asbestos that you've done have been published in peer reviewed journals?
A Risk assessment has many steps. I would say the work that I did at EPA passes more scrutiny than the several -- in our journal we have three to four peer reviewers. So the work I did at EPA was certainly carefully scrutinized. I did the risk assessment work on the first issue of Ingestion of Asbestos over a period of time in the 70's, from 73 to 78, to determine whether or not amosite from reserved mining might be a

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causative agent of any health issues for people who consumed the water from Lake Superior.

In 1978, I was co-author of the first risk assessment work for EPA's air programs. In 19 -- late 84, I believe, is the date, I was responsible for commissioning the work with Dr. Nicholson to do the cancer chapter for the 1986 publication of EPA's risk assessment.

MR. MULLADY: Excuse me, Your Honor, but --

THE WITNESS: I'm getting there.

MR. MULLADY: Excuse me, ma'am. I believe the question, Your Honor, was very simply, have any of her risk assessments been published in a peer reviewed journal.

THE WITNESS: I'm getting there. I said these --

MR. MULLADY: Well, that was the question.

THE WITNESS: -- these risk assessments that I'm a part of received far more scrutiny than just a risk assessment journal. So, to dismiss them is not appropriate. But in my resume, you will see that I published one of the earliest papers, if not the earliest papers, inspecting a part of the risk assessment process which is the proposed six different mechanisms of action. Very critical to both the hazard identification and to exposure in dose response.

Later, within the next year, I published comparative risk assessment investigating the risk associated with asbestos in place and asbestos removal. So, it was a comparative risk

assessment. So, those two --

Q And, what peer reviewed journal was that published in, ma'am?

A Pardon. Yes --

MR. BERNICK: Excuse me.

MR. MULLADY: No --

THE WITNESS: It's on my resume.

MR. BERNICK: Excuse me. She said she can answer the question. She's already given you two examples, we're now onto the third. If you want to ask what journal it's been published in, Your Honor, she can -- that's a follow up question. He shouldn't be interrupting the witness.

THE COURT: I think you did ask, so I think she should be entitled to answer the question.

Q I'm sorry, ma'am, have you finished your answer?

A Yes. I was just citing these publications. They're on my resume and they're peer reviewed journals and they were some of the first work done in risk assessment on asbestos. Both on mechanisms of action, one of the very important questions to risk assessment, and one on full comparative risk assessments in particular circumstances on asbestos, exposures and disease.

Q I asked you the same -- well, my partner, Garret Rasmussen or Mr. Slocum, I can't remember which, asked you this same question at your deposition. Can we take a look at Pages 27 to 28, please. Let's go back up a little bit further in the

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text.

A I was speaking to the EPA risk assessments.

Q Excuse me, ma'am, I haven't asked you a question yet.

A That they were not published -- well, you put this in front of me.

Q I'm about to ask you a question and then I'll be happy to listen to your answer. At deposition, were asked the following question: "Do any of your publications attempt to assess whether exposure to asbestos caused an individual's illness?" And your answer was: "I have worked with asbestos and risk assessment since the earliest days at EPA starting in the 1970's. One does not generally publish a risk assessment and I don't think I have attempted to publish any of my risk assessments that have dealt with asbestos." I'm sorry, the question was -- and let's go further in, so we see the full context here. The question is a publication that addresses whether exposure to asbestos caused an individual's illness as opposed to assessments of the risk to a general population. And you asked, "When you say published, what do you mean?" And, the question was: "Let's start out, published in a peer reviewed journal?" And your answer was: "No, I don't think I have published any of my risk assessments in a peer reviewed journal. All of my risk assessments addressed risk to individuals as well as risk most all, risk to individuals as well as the risk to populations." Was that the testimony that

you gave in deposition, ma'am?

A Yes, and he was asking me about the EPA risk assessments and I was explaining that those risk assessments normally are not published in peer reviewed journals. Today I'm saying, those risk assessments are under more scrutiny than articles in peer reviewed journals because they are read by so many different parties and scrutinized by so many scientists.

Q Thank you, ma'am.

A And, he has my resume, so he certainly knew my publications otherwise.

MR. MULLADY: I think we understand your answer. Thank you, ma'am. No objection to the proffer.

MR. BERNICK: Nate? I take it from Mr. Finch's shaking his head that you don't -- the ACC does not have an objection.

MR. FINCH: No, objection to the proffer.

MR. BERNICK: Thank you.

THE COURT: All right. The witness may express an expert opinion as proffered.

CONTINUED DIRECT EXAMINATION

BY MR. BERNICK:

Q Dr. Anderson, could you just tell us and we're going to get right to the risk assessment that we're going to be talking about in this case here, on the very next questions, but could you just tell us what the basic elements of a risk assessment

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are as a general matter, and I'd like to show demonstrative 2268.

A Yes. I think as I said earlier, the first question is, can an agent cause disease and, of course, we know asbestos in certain situations considering fiber size and type, can absolutely cause disease. The dose response assessment is the next critical step, that's been discussed, I think here. Exposure assessment is then the evaluation of the cumulative exposure that individuals and populations receive and the fourth step is the characterization, wedding all the previous three steps together to address the essential question and in this case, are exposures to Grace product groups responsible for claimants disease.

Q Thank you. Now, we've already heard from Dr. Lees. Did he participate in the risk assessment that was associated, or that's been done for Grace in this case?

A Yes, he did.

Q Okay. And just indicate for the Court where you're going generally, what role, if any, did you play, what was the nature of your role in connection with the risk assessment that was done for purposes of this case?

A My role, I suppose is, I integrated the information from the first part of exposure assessment, the concentration data from the industrial hygiene studies I received from Dr. Lees. I added the frequency and duration portion of the exposure

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assessment to arrive at a cumulative exposure. Dr. Moolgavkar has discussed, I believe the dose response characteristics for asbestos and has identified certain levels that I've called benchmarks, so I could have the benchmarks for the risk characterization. I have then gone forward to evaluate the cumulative exposures for groupings, we call nature of exposure groups and then compared their exposures to Dr. Moolgavkar's benchmarks.

I've then organized the outcome of that comparison to pass that outcome on for the next analysis which I understand is Dr. Florence's analysis.

Q Thank you. I want to show you what we have as a demonstrative, and we've put it in the form of a big magnetic board, Your Honor, we will also tender the demonstrative as a slide or a smaller version of this after it's completed, as 2296, for its files.

UNIDENTIFIED MALE SPEAKER: What was the number for that, Dave?

MR. BERNICK: 2296.

Q But, Dr. Anderson, looking to the portion of 2296 that's displayed on the magnetic board, does this show the sequence of, or the different steps of the risk assessment that you participated in in connection with this case?

A Yes, it does. As I said, the concentration information for the exposed claimants groupings was provided, analyzed and

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provided to me by Dr. Lees. I performed the -- I completed the exposure assessment by using his concentration values. I added the exposure frequency and duration to arrive at cumulative exposures of dose and then I compared those cumulative exposures of dose to benchmarks from Dr. Moolgavkar's work and from the literature that he used to define benchmarks for comparison.

Q Okay. This formula that appears at the top of 2296, concentration times exposure frequency times duration equals dose, and then to proceed to look at dose response and then to risk, is that a sequence and a formula that is unique to this case?

A No, it is not. This has been the sequence and formula, if you will, that has been consistently used since the origins of risk assessment in my background since 1976, codified in the four steps by the National Academy in 1983.

Q Okay. Let's begin with the first part of that formula, which is dose. The calculation of dose. Turning to Exhibit 2270, could you explain for us the central question that was addressed in the risk assessment for this case, as concerns the calculation of dose?

A Yes. The central question is how our accepted methods, used to calculate the doses resulting from exposure to Grace products and the ingredients of that analysis are listed here, the concentration, multiplied by the frequency, by the duration

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of exposures, provide the cumulative exposure for the individual or the claimant group.

A Okay. Now, we've already heard from Dr. Lees but I want to talk first of all about the concentration part of it and ask you just to give the Court an overview of what it is that Dr. Lees did as you understood it, in order to provide a foundation for how it is that you then pick up from his work and what you did with it. So could you just give the Court a very brief overview of your understanding of what it is that Dr. Lees did?

A Well, Dr. Less did several things. The first thing he did is, he defined or he flushed out the exposure definitions for the nature of exposure categories A through E.

Q Okay. Let's show 2269 and I want to ask you whether 2269 reflects the basic categories that Dr. Lees worked with?

A It does and the Category A is the category of individuals who mixed Grace products. Category B, category of individuals who were in areas to exercise their trade by removing or cutting Grace products. The C category is the category of individuals involved in installing Grace products. D is a person at a site where these products were being used, but they were not in the room with, they were out of the line of sight, most likely with the products in it. Category E would be an individual in the space when the products were being either applied or sprayed.

Q Now, in this chart it emphasizes that these are all job

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activities that were analyzed as concerns Grace products. For purposes of the work, the risk assessment that was done in this case, did you or others working with you analyze the exposures that the claimants had with respect to -- or industrial hygiene data that was available with respect to job activities relating to non-Grace products?

A No. We did not have information relating to non-Grace products. So, we were focused on what exposure did these particular claimants have from Grace products, per se.

Q Okay. Now, as an example, Dr. Lees talked about the fact that when it came to the personal installation we have a person who is spraying, what kind of product that Grace was being analyzed when it came to the asbestos containing mixture being sprayed from a hose?

A Grace had fireproofing products that were being sprayed and I understand from Dr. Lees that they were using a wet method of application for their products.

Q That's exactly what I was going to get to. In connection with the work that was done to do the risk assessment in this case, did you all focus not on the wet application, not only on the wet application pertaining to Grace product, but did you also analyze dry applications of other kinds of products?

A Dr. Lees is aware of the difference between the dry applications, which were not Grace products, as I understand it and the wet application of Grace products. We also analyzed or

he analyzed the exposure data and we used that to analyze further through the exposure duration and frequency the exposures to individuals who were painting on or troweling on Grace products.

Q Okay. But when you carried through the industrial hygiene data, was that the data that related to the wet application or the dry application done by others?

A His work was related to the Grace installation per se. The wet application.

Q Okay. Now, what kinds of values, what kinds of values did Dr. Lees calculate after having reviewed the industrial hygiene data with respect to these different job activities? What kind of calculations did he do?

A Well, he first of all defined the exposure for the categories. He gathered the data, he qualified the data and he provided his time weighted average mean concentrations by two analyses. One was what he called a stratified analysis where he took each study and averaged the study to get the mean of the study and he averaged them across the studies and the other was what is, I think he referred to as a meta analysis when he averaged all the values across all the studies. So, he provided all of this to me.

Q Okay. Now, you just reused the word mean, in talking about a mean concentration, and mean also appears on the overall exhibit, which is 2269. Let's deal with this question

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of the mean concentration. Tell us whether or not industrial hygiene data that is generally available in risk assessment, tell us whether or not industrial hygiene data reflects to varying degrees, variability, variability.

A Well, certainly it does because industrial hygienists collect their data on different days and different places when environmental factors are changing, and there are variations in those factors. There will be some other variations in collection methods and analytical methods and even in the way some individuals work with the products.

Q Showing you 2271, does this capture some of the reasons why or the drivers for variations, and what kinds of affects they can have on concentration values.

MR. MULLADY: Objection, leading.

MR. BERNICK: That's fine.

Q Could you explain, please, Dr. Anderson, what it is that Exhibit 2271, the demonstrative, reflects as concerns variation?

MR. MULLADY: Objection, leading.

THE COURT: No, it is not leading. Explain what this slide reflects. It is leading. If anything. You can correct the statement, thank you, not leading.

Q Go ahead, Dr. Anderson.

THE COURT: You can answer.

THE WITNESS: May I?

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THE COURT: Yes, please.

THE WITNESS: Well, I think the question is, why is that variation measure data and we've always dealt with this. We dealt with this very overtly at EPA in the early years when we were trying to measure for the first time environmental data and use it for exposure assessment. We quickly found that we had variation in sample technique, analytical technique, different people may apply or do something as a receptor that's different from another person. And we found that environmental variables were very essential. Which way the wind is blowing and where the samples are taken, with respect to humidity, and open windows, it's very difficult to get just samples that are not widely influenced by these factors.

At the end of the day, I know that Dr. Lees has stated that the environmental variables predominate by far. From my experience I would agree with him, that's what I've seen in the data that I've seen collected over the last 30 years.

Q Now, Exhibit 2269 reflects that in the work that was done on concentration, the concentration data and I think you've indicated the same thing, focused on -- a mean was calculated for the concentration data, is that accurate?

A That is accurate.

Q Okay. Now, tell us why it is that the mean is something that is of value, or that is the step that we're talking about

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